

In the Specification:

At page 1, line 10, please insert the following paragraph.

A1
~~This invention was made with Government support under Grant No. ROIGM60396-01, awarded by the National Institutes of Health and Grant No. MCB-9506191. The Government has certain rights in this invention.~~

Please replace the paragraph beginning at page 22, line 15 with the following:

A2
~~Due to the basal regulation of anion and K⁺ channels in *abh1* without~~
addition of exogenous ABA, experiments were pursued to analyze whether mechanisms lying further upstream confer ABA hypersensitivity in *abh1*. Anion channels are activated and inward-rectifying K⁺ channels are down-regulated by upstream [Ca²⁺]_{cyt} elevations (J.I. Schroeder & S. Hagiwara, *Nature*, 338:427 (1989)). Therefore we directly investigated whether *abh1* modulates ABA-induced [Ca²⁺]_{cyt} elevations in time-resolved cameleon [Ca²⁺]_{cyt} imaging experiments (G. J. Allen *et al.*, *The Plant J.*, 19:735 (1999)). Stomata were opened by exposing plants for 12 hours to 95% humidity. In wild-type, 56 % (n=32 of 57) of guard cells showed no [Ca²⁺]_{cyt} increase in response to a low concentration of 0.5 μM ABA and the remaining 44% (n=25) cells typically showed only one [Ca²⁺]_{cyt} increase with an average peak increase of 170 ± 25 nM [Ca²⁺]_{cyt}. Interestingly, in *abh1* guard cells, 0.5 μM ABA elicited [Ca²⁺]_{cyt} increases in 64% of guard cells (n= 41 of 64 cells) with a larger average peak increase of 280 ± 22 μM. Only 19% of the cells (n=12) responded with one [Ca²⁺]_{cyt} elevation while 45% of *abh1* cells (n=29) showed multiple repetitive [Ca²⁺]_{cyt} increases at 0.5 μM ABA. Only 36% of *abh1* cells (n=23) showed no response to 0.5 μM ABA. Statistical analyses of responsive versus non-responsive cells confirmed that the ABA responsiveness of *abh1* guard cells was significantly enhanced ($\chi^2=4.96$, P<0.03). Furthermore both the number of [Ca²⁺]_{cyt}

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transients per cell ($P < 0.001$) and their amplitudes ($P < 0.01$) were significantly larger in *abh1* than in wild-type. $[Ca^{2+}]_{cyt}$ imaging analyses and stomatal aperture measurements demonstrate that the *abh1* mutation enhances early ABA signaling mechanisms upstream of ABA-induced $[Ca^{2+}]_{cyt}$ elevations.

Please replace the paragraph beginning at page 23, line 17 with the following:

A3

The *ABH1* gene was identified by plasmid rescue and the corresponding cDNA (2547 bp) was isolated. Briefly, a 278 bp genomic fragment adjacent to the right border of the T-DNA insertion was isolated from *abh1* plants using plasmid rescue as follows: 5 μ g of genomic DNA was digested with *Hind*III, self-ligated and transformed into *E. coli* ElectroMAX DH12S (GibcoBRL, Lifetechnology). Plasmid extracted from cells growing on carbenicillin was sequenced. Primers were then generated to amplify 5316 bp genomic DNA flanking the rescued sequence (GenomeWalker Kit, Clontech). A 8248bp *Cla*I genomic fragment containing the full *ABH1* locus was cloned from BAC T10F2 (*Arabidopsis* Biological Research Center) into the plant expression vector pRD400. *ABH1* coding sequences were amplified from an *Arabidopsis* Columbia leaf cDNA library by rapid amplification of cDNA ends (RACE PCR, Marathon cDNA Amplification Kit, Clontech) using the plasmid rescue sequence internal primer (5' GAAGCTCAACTCGTTGCTGGAAAG 3'; SEQ ID NO:4) and its reverse. The total cDNA of 2547 bp was then amplified using *pfu* DNA polymerase (Stratagene), cloned in pMON530 and sequenced. *ABH1* 5' UTR (1250bp) was amplified from genomic DNA by PCR using *pfu* DNA polymerase and subcloned in pCAMBIA1303 (Genbank AF23299) containing a promoterless glucuronidase reporter gene. All sequences amplified by PCR were checked by sequencing (Retrogen, CA).

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Please cancel the present informal "SEQUENCE LISTING", pages 30 and 31, and insert therefor the accompanying paper copy of the Sequence Listing, page numbers 1 to 5, at the end of the application.